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Weight Loss after Bariatric Surgery and Periodontal Changes: A 12-Month Prospective Study

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Running title: weight loss surgery and periodontal disease

ABSTRACT

Background: Several longitudinal studies have explored the association of obesity and weight gain with periodontal disease. However, the effect of weight loss on periodontal tissues remains unclear.

Objective: To explore whether weight loss after bariatric surgery was associated with changes in periodontal measures over 12 months.

Setting: Two public hospitals in São Paulo, Brazil.

Methods: We used data from 110 morbidly obese patients (BMI>40 kg/m^2 or ≥35 kg/m^2 with comorbid conditions) who underwent bariatric surgery between April 2011 and March 2013. Data on demographic factors, body mass index (BMI), smoking habits and glucose levels were extracted from medical records pre-operatively and after 6 and 12 months post-surgery. A full-mouth periodontal examination was conducted by trained examiners to assess probing pocket depth (PPD), clinical attachment loss (CAL) and bleeding on probing (BOP) at baseline, 6 and 12 months. Data were analyzed using linear mixed effects (LME) models.

Results: BMI was not significantly related to the proportion of sites with BOP at baseline, but it was negatively associated with the rate of change in the proportion of sites with BOP. The greater the BMI loss the higher the proportion of sites with BOP, particularly 6 months after surgery. However, BMI was not associated with baseline PPD and CAL or rates of changes in these periodontal outcomes.

Conclusion: The findings suggest that weight loss was associated with increased gingival bleeding, showing a peak at 6 months after bariatric surgery. Periodontal pocketing and attachment loss remained unchanged during the study period.

Keywords: body weight; bariatric surgery; periodontal diseases; cohort studies; adults

INTRODUCTION

The prevalence of obesity has increased epidemically in both developed and developing countries over the last few decades [1]. Obesity is a metabolic condition caused by an energy imbalance (i.e. when energy intake exceeds energy expenditure), which subsequently leads to an increase in adipose tissue deposits [2]. As adipocytes exert a number of endocrine functions [3], increased adiposity is associated with a state of low-grade inflammation and insulin resistance [2, 4]. This obesity-related pro-inflammatory status appears to be involved in cardiovascular diseases, type 2 diabetes, metabolic disorders and certain cancers [5, 6].

Evidence is growing for a possible link between obesity and periodontal disease [7-9]. Obesity related inflammation may promote periodontitis by secretion of inflammatory markers by the adipose tissue that may increase gingival inflammation and promote bacterial proliferation on the tooth root surface [10, 11]. A recent review has also reported that weight gain was associated with incidence of periodontitis, although only 5 studies were identified [12]. Since the unfavorable inflammatory profile associated with increased adiposity can be improved during a period of weight loss [13], what is missing in the literature is an evaluation of the effect of weight loss on periodontal tissues.

Several methods have been proposed for weight loss in obese patients, like dieting and physical exercise, pharmacological treatment, and surgical intervention [14]. Bariatric surgery is an effective therapy for the treatment of obesity compared with non-surgical interventions [15]. The benefits of bariatric surgery include significant and durable weight loss, improved or remission of obesity related comorbidities, and improved quality of life [15, 16]. Limited evidence, mainly coming from case reports and cross-sectional studies, suggests that higher levels of dental caries, periodontal diseases and tooth wear may be found in patients after bariatric surgery [17, 18]. In a recent prospective study, a resolution of systemic inflammation after bariatric surgery –i.e. significant decreases in C-reactive protein (CRP)

and glucose levels after surgery– did not seem to affect the course of periodontal disease. On the contrary, the mean periodontal pocket depth and attachment loss increased significantly 6 months after bariatric surgery [19, 20].

The purpose of this study was to explore whether weight loss after bariatric surgery was associated with changes in periodontal measures over 12 months.

METHODS

This report adheres to the Strengthening the Reporting of Observational Studies (STROBE) guidelines [21].

Participants

A total of 150 morbidly obese patients (BMI>40.00 Kg/m^2 or ≥35.00 Kg/m^2 with comorbid conditions) were recruited from the patient pool receiving bariatric surgery (Roux-en-Y) in two public hospitals in São Paulo, Brazil, between April 2011 and March 2013. Patients with history of any infectious diseases, those who were pregnant or breastfeeding, using anti-inflammatory agents or antibiotics 3 months prior to the study and those who have fewer than six teeth were excluded from the study.

The study protocol was approved by the Research Ethics Committees of the two Medical Schools Hospitals (Ref: 315/08 and 468/08). All patients were informed of the purpose of the investigation and signed a written informed consent before voluntary participation.

After exclusions, there were 110 patients (aged 20 to 60 years at baseline) who had periodontal data in at least two of the three examinations (baseline plus 6 or 12 months). Periodontal data were available for 110, 90 and 110 participants preoperatively and at 6 and 12 months after surgery, respectively. That means 90 patients (82%) contributed to all three waves of periodontal data whereas the rest (18%) to two waves. A post-hoc calculation showed that this sample size had a 90% power to identify an 8%-difference in the proportion

of sites with bleeding on probing (BOP) before and after surgery, with standard deviation of 25% in each measurement occasion and a correlation of 0.50 between measurements.

Data collection

Data were collected from medical records and through clinical examinations. Demographic factors (sex and age), anthropometric measures (height and weight), smoking habits, and fasting blood glucose (FBG) levels were extracted from patients' medical records preoperatively and 6 and 12 months after surgery. In line with local protocols, an FBG greater than 100 mg/dL was used for diagnosis of diabetes. Weight loss after bariatric surgery was expressed as change in body mass index (BMI). Patients did not receive any dental care or oral health advice during the hospitalization period.

Participants were also invited to a periodontal examination at baseline and each control visit. A North Carolina periodontal probe was used for the clinical inspection of all present teeth, excluding third molars. A full-mouth examination protocol was used, inspecting six sites per tooth (mesio-facial, mid-facial, disto-facial, mesio-lingual, mid-lingual, and disto-lingual) to measure probing pocket depth (PPD), clinical attachment loss (CAL) and bleeding on probing (BOP). PPD was the distance from the gingival margin to the base of the pocket whereas CAL was the distance between the cement-enamel junction and the base of the pocket. Three periodontal outcome measures were evaluated, namely the mean PPD and CAL across all examined sites and the proportion of sites with BOP. Examinations at three times, namely pre-operatively, 6 and 12 months after bariatric surgery were performed by two calibrated dentists, with Kappa values for intra- and inter-examiner reliability of 0.82 and 0.80, respectively.

Statistical analysis

Linear mixed effects (LME) models were used to estimate the longitudinal association between change in BMI and changes in BOP, PPD and CAL over 12 months. LME models use all available outcome data over the follow-up period, handle unequally spaced observations over time and take into account the fact that repeated measures on the same individual are correlated [22, 23]. All analyses were run in Stata Statistical Software (Release 13. College Station, TX: StataCorp LP) using the *mixed* command.

We explored the association of BMI with BOP, PPD and CAL in separate set of models. We fitted both the intercept and the slope with time as random effects, allowing for individual differences in periodontal measures at baseline and rates of change in periodontal measures over the follow-up period. Survey waves (0, 6 and 12 months coded as 0, 1 and 2 respectively) were used as the underlying time scale in all models (fitted as a categorical time indicator). First we estimated a model without any covariates (null model) to establish the rate of change in BOP within the observed period. Next, we tested the effect of BMI (for every 10-unit change) on baseline BOP levels controlling for sex, age, smoking status and diabetes. We then tested the association of BMI with changes in BOP over time by adding the interaction between BMI and the time indicator to the main effects model. The main effect for BMI estimates the effect on BOP at baseline whereas the interaction term between BMI and time estimates the effect of BMI on change in BOP over 12 months. We presented significant associations with changes in BOP using line graphs to aid interpretation. The same steps were followed when testing the association of BMI with PPD and CAL, respectively. As smoking causes periodontal vasoconstriction [24], we tested whether the effect of BMI on periodontal measures was different in smokers and non-smokers by testing the significance of the statistical interaction between BMI and smoking status when added to the model.

RESULTS

A total of 110 adult patients (88% women), with a mean age of 38.5 years (Standard Deviation [SD]: 9.8, range: 20 to 60) were included in this study. The baseline characteristics of the sample are shown in Table 1. The mean proportion of sites with BOP was 24.6% at baseline (SD: 23.4; range: 0-100) whereas the mean PPD and CAL were 1.77 mm (SD: 0.47; range: 1.01-3.10) and 1.86 mm (SD: 0.60; range: 0.58-4.24), respectively.

Larger variations were observed for BOP (from 24.6% at baseline to 32.0% at 6 months to 30.8% at 12 months) than for mean PPD (from 1.77mm at baseline to 1.74mm at 6 months to 1.70mm at 12 months) or mean CAL (from 1.86mm at baseline to 1.89mm at 6 months to 1.88mm at 12 months) after bariatric surgery (Figure 1). BMI was not significantly related to the proportion of sites with BOP at baseline (Table 2), but it was negatively associated with the rate of change in the proportion of sites with BOP ($p<0.05$ for the interaction between weight and the time indicator). The greater the BMI the higher the proportion of sites with BOP, particularly at 6 months after surgery. This trend is shown in Figure 2 for different levels of BMI loss. On the other hand, BMI was not associated with baseline PPD and CAL or the rates of changes in these periodontal outcomes. Age and diabetes were the only factors associated with mean PPD and CAL at baseline (Table 2). The interaction term between BMI and smoking status was not statistically significant ($p>0.05$).

DISCUSSION

This study shows that weight loss after bariatric surgery was associated with an increase in the proportion of sites with gingival bleeding, over and above the independent effects of demographic factors, smoking status and diabetes. Only minor changes in periodontal pocket depth and attachment loss were noted, which were not associated with weight loss.

The present results should be interpreted keeping in mind some study limitations. First, this study was based on a convenience sample of obese adults, and as such, the results are not generalizable beyond this group of participants. Second, the inclusion of a control group (morbidly obese patients that did not undergo bariatric surgery) running in parallel to the intervention would have strengthened the study design. That said, a control group is particularly useful to clarify whether improvements in outcome measures (which were not observed in this study) are due to the intervention being tested. Third, anthropometric measurements were not purposefully collected for this study but extracted from medical records. Although this may raise concerns about increased measurement error, there is evidence that body weights documented in medical records are exchangeable with body weights recorded in a research setting [25, 26], particularly among women –who represented 88% of our study group– [27, 28]. Fourth, our regression models did not include a control variable for socioeconomic position, which is strongly related to both obesity [29] and periodontal disease [30]. Data on socioeconomic circumstances were not routinely collected as part of patients' medical records. However, restricting the sample to two neighboring hospitals serving the same population provided a control for confounders during the study design as all participants were exposed to similar social and environmental circumstances. Information on factors such as feeding practices and oral hygiene were not collected either. This omission does not affect the overall estimate for the association between weight loss and periodontal disease because the above factors are considered merely intermediates (not confounders) of the hypothesized association. However, they would have helped clarifying why weight loss would be associated with increased gingival bleeding.

The present findings suggest that weight loss was related to increased gingival bleeding but not to periodontal pocketing or attachment loss, offering no support for the hypothesized inflammatory pathway linking obesity to periodontal disease. If obesity and periodontal

disease are causally related, a reduction in body weight should improve periodontal conditions, at least in terms of signs of gingival inflammation. Improvements in other common periodontal indicators, such as pocket depth and loss of attachment, may be more difficult to prove since they may require periodontal treatment after all. Weight loss does improve inflammation in terms of obesity-related inflammatory markers, specifically characterized by a decrease in inflammatory markers (CRP, tumor necrosis factor-α, interleukin-6 and leptin) and an increase in the anti-inflammatory marker, adiponectin [13, 31, 32]. However, it is also worth noticing that the evidence suggests that at least 2 years are required post-surgery for stabilization of physiological processes and the inflammatory profile [13]. Thus, it is possible that we were not able to identify changes in other periodontal measures because of the short follow-up period.

An alternative explanation for these findings has to do with the nutritional and anatomic changes after bariatric surgery, which may increase the risk of oral complications [17]. Bariatric surgery causes a reduction of gastric capacity and consequently a decrease in food ingestion [15]. These changes lead to new feeding patterns post-surgery, including frequent small meals –i.e. grazing– and soft foods [16] that adhere to the tooth surface throughout the day [20]. This is in addition to a recent report of an increase in *Porphyromonas gingivalis* 6 months after bariatric surgery [19], suggesting that changes in the amount and microbial composition of the dental biofilm are a common occurrence among bariatric patients. Gastroesophageal reflux and vomiting are other common side-effects of bariatric surgery [33], which may result in erosive lesions of the oral mucosa, including gingival tissues [17]. Finally, bariatric surgery may lead to restriction and/or malabsorption of nutrients, causing deficiencies in iron, calcium, folate, and vitamins B12, A, D, E and K, some of which are important to maintain periodontal health [34].

This study has some implications for practice and further research. Health professionals should be aware of possible oral complications of bariatric surgery. Dentists should be part of the multidisciplinary team taking care of patients undergoing weight loss surgery in order to monitor their periodontal status throughout the entire process, paying particular attention to the first 6 months after surgery. From a research perspective, future prospective studies should explore the long-term effects of weight loss on periodontal conditions and the interrelationship between weight loss, obesity-related inflammatory markers and periodontal disease. The role of local factors (such as feeding practices and oral hygiene) in the relationship between weight loss after surgery and periodontal disease should be assessed.

CONCLUSION

This prospective study shows that weight loss was associated with increased gingival bleeding, showing a peak at 6 months after bariatric surgery. Periodontal pocketing and attachment loss remained unchanged during the first 12 months post-surgery.

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DISCLOSURES

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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Table 1. BMI at baseline and 6 and 12 months post-surgery, by baseline characteristics

Baseline characteristics	n	%	Time 0 (n=110)		Time 1 (n=90)		Time 2 (n=110)	
			Mean	(SD)	Mean	(SD)	Mean	(SD)
Sex								
Men	13	11.8	50.5	(8.6)	37.0	(7.9)	32.4	(4.6)
Women	97	88.2	49.7	(8.4)	35.7	(6.0)	32.3	(6.2)
P value[a]			*0.76*		*0.52*		*0.95*	
Age group								
20-29 years	17	15.5	49.4	(6.7)	35.0	(5.8)	31.0	(5.0)
30-39 years	48	43.6	51.4	(10.3)	37.1	(7.7)	32.9	(7.6)
40-49 years	25	22.7	49.2	(6.0)	35.6	(3.9)	32.3	(4.2)
50-60 years	20	18.2	47.1	(6.1)	33.8	(4.4)	32.2	(4.3)
P value[a]			*0.26*		*0.31*		*0.71*	
Smoking status								
Non-smoker	87	79.1	49.9	(8.7)	35.9	(6.3)	32.6	(6.3)
Smoker	23	20.9	49.5	(7.2)	35.6	(6.2)	31.4	(4.8)
P value[a]			*0.85*		*0.84*		*0.39*	
Diabetes								
No	83	75.5	50.8	(9.0)	36.3	(6.8)	32.9	(6.6)
Yes	27	24.6	46.7	(5.0)	34.7	(4.1)	30.8	(3.4)
P value[a]			*0.03*		*0.29*		*0.12*	

[a] t-test was used in all comparisons except for age groups where analysis of variance was used

Table 2. Linear mixed models for the association between baseline BMI and periodontal measures over 12 months

Baseline characteristics	% sites with BOP			Mean PPD (mm)			Mean CAL (mm)		
	Coef.	**[95% CI]**	**p value**	**Coef.**	**[95% CI]**	**p value**	**Coef.**	**[95% CI]**	**p value**
BMI[a]	0.3	[-4.7, 5.3]	0.90	0.1	[-0.0, 0.2]	0.13	0.1	[-0.0, 0.2]	0.13
Time indicator									
Time 1	0.0	[Reference]		0.0	[Reference]		0.0	[Reference]	
Time 2	15.8	[-11.9, 43.4]	0.26	1.9	[1.3, 2.4]	<0.01	2.1	[1.5, 2.8]	<0.01
Time 3	11.1	[-17.5, 39.8]	0.45	1.9	[1.3, 2.4]	<0.01	2.2	[1.6, 2.9]	<0.01
BMI X Time 2	-7.7	[-15.4, -0.2]	0.04	0.0	[-0.1, 0.2]	0.76	0.1	[-0.1, 0.2]	0.43
BMI X Time 3	-8.2	[-17.7, 1.4]	0.09	0.0	[-0.1, 0.2]	0.81	0.1	[-0.1, 0.3]	0.29
Sex									
Men	0.0	[Reference]		0.0	[Reference]		0.00	[Reference]	
Women	5.1	[-7.4, 17.7]	0.42	-0.1	[-0.3, 0.2]	0.64	-0.2	[-0.5, 0.1]	0.32
Age group									
20-29 years	0.0	[Reference]		0.0	[Reference]		0.0	[Reference]	
30-39 years	1.9	[-10.4, 14.2]	0.76	0.2	[-0.1, 0.4]	0.15	0.2	[-0.1, 0.5]	0.27
40-49 years	4.5	[-8.9, 18.0]	0.51	0.2	[-0.0, 0.5]	0.10	0.4	[0.1, 0.7]	0.02
50-60 years	5.7	[-8.6, 20.0]	0.43	0.3	[0.0, 0.6]	0.03	0.5	[0.2, 0.9]	<0.01
Smoking status									
Non-smoker	0.0	[Reference]		0.0	[Reference]		0.0	[Reference]	
Smoker	4.1	[-5.7, 13.8]	0.42	-0.1	[-0.3, 0.1]	0.56	-0.1	[-0.3, 0.2]	0.53
Diabetes									
No	0.0	[Reference]		0.0	[Reference]		0.00	[Reference]	
Yes	-0.6	[-8.2, 7.1]	0.89	0.2	[0.0, 0.3]	0.02	0.2	[0.1, 0.4]	0.01
Intercept	11.0	[-16.8, 38.7]	0.44	1.6	[1.1, 2.1]	<0.01	1.8	[1.1, 2.4]	<0.01

BOP: Bleeding on probing; PPD: probing pocket depth; CAL: clinical attachment loss
BMI was assessed per every 10-unit change